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PCTFULL, DISSABS' ENTERED AT 18:56:54 ON 09 NOV 2004

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L2	10 DUP REM L1 (9 DUPLICATES REMOVED)
L3	808 S SEMI-ALLOGENEIC
L4	88 S L3 AND ANTIGEN (A) PRESENTING
L5	70 DUP REM L4 (18 DUPLICATES REMOVED)
L6	35 S L5 AND SYNGENEIC

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L1: Entry 1 of 1

File: USPT

Feb 13, 2001

US-PAT-NO: 6187307

DOCUMENT-IDENTIFIER: US 6187307 B1

TITLE: Cancer immunotherapy with semi-allogeneic cells

DATE-ISSUED: February 13, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Cohen; Edward P.	Chicago	IL		

US-CL-CURRENT: 424/93.21; 424/93.71, 435/325, 435/366, 435/372, 435/455, 536/23.5

CLAIMS:

What is claimed is:

1. A semi-allogeneic immunogenic cell for administration to an animal recipient, which comprises an antigen-presenting cell expressing at least one class I MHC or class II MHC determinant that is syngeneic to the recipient and at least one class I or class II MHC determinant that is allogeneic to the recipient, wherein said antigen presenting cell is transformed with and expresses DNA coding for at least one antigen, and wherein said antigen or a part thereof, when complexed with said MHC class I or class II determinant at the cell surface, is recognized by T cells.
2. A semi-allogeneic immunogenic cell for administration to an animal recipient, which comprises an antigen-presenting cell expressing at least one class I MHC or class II MHC determinant that is syngeneic to the recipient and at least one class I or class II MHC determinant that is allogeneic to the recipient and wherein said antigen presenting cell is transformed with and expresses DNA isolated from a neoplasm or a tumor of the recipient.
3. The semi-allogeneic immunogenic cell of claim 1 or 2, wherein said antigen presenting cell is further transformed with a coding sequence for at least one cytokine.
4. The semi-allogeneic immunogenic cell of claim 3 wherein the cytokine is selected from the group consisting of interleukin-1, interleukin-2, interleukin-3, interleukin-4, interleukin-5, interleukin-6, interleukin-7, interleukin-8, interleukin-9, interleukin-10, interleukin-11, interleukin-12, interferon-.alpha., interferon-.gamma., tumor necrosis factor, granulocyte macrophage colony stimulating factor, and granulocyte colony stimulating factor.
5. The semi-allogeneic immunogenic cell of claim 1 or 2, wherein the antigen-presenting cell is selected from the group consisting of a fibroblast, a

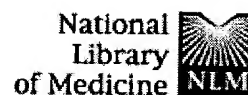
macrophage, a B cell, and a dendritic cell.

6. The semi-allogeneic immunogenic cell of claim 2, wherein the neoplasm is selected from the group consisting of melanoma, lymphoma, plasmocytoma, sarcoma, glioma, thymoma, leukemias, breast cancer, prostate cancer, colon cancer, esophageal cancer, brain cancer, lung cancer, ovary cancer, cervical cancer, and hepatoma.
7. The semi-allogeneic immunogenic cell of claim 2 wherein the DNA isolated from a neoplasm or tumor comprises coding sequences for tumor associated antigens.
8. The semi-allogeneic immunogenic cell of claim 2 wherein the DNA isolated from neoplastic cells comprises coding sequences for tumor associated antigens that are associated with a tumor, wherein said tumor is selected from the group consisting of melanoma, lymphoma, plasmocytoma, sarcoma, glioma, thymoma, leukemias, breast cancer, prostate cancer, colon cancer, esophageal cancer, brain cancer, lung cancer, ovary cancer, cervical cancer, and hepatoma.
9. A therapeutic composition comprising the semi-allogeneic immunogenic cell of at least one of claims 1, 2, 7, or 8 admixed with a therapeutically acceptable carrier.
10. A therapeutic composition comprising the semi-allogeneic immunogenic cell of claim 3 admixed with a therapeutically acceptable carrier.
11. A therapeutic composition comprising the semi-allogeneic immunogenic cell of claim 4 admixed with a therapeutically acceptable carrier.
12. A therapeutic composition comprising the semi-allogeneic immunogenic cell of claim 5 admixed with a therapeutically acceptable carrier.
13. A therapeutic composition comprising the semi-allogeneic immunogenic cell of claim 6 admixed with a therapeutically acceptable carrier.
14. A semi-allogeneic immunogenic cell for administration to an animal recipient, which comprises an antigen-presenting cell expressing at least one of class I or class II MHC determinants, wherein said antigen presenting cell is genetically selected such that at least one of said class I MHC or class II MHC determinants is syngeneic to the recipient and at least one of said class I or class II MHC determinants is allogeneic to the recipient, wherein said antigen presenting cell expresses at least one antigen, and wherein said antigen or a part thereof, when complexed with said MHC class I or class II determinant at the cell surface, is recognized by T cells.

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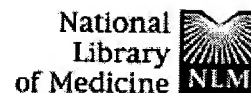
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#56	Search #54 AND semiallogeneic	18:42:48	4
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#48	Related Articles for PubMed (Select 9439644)	18:37:19	186
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#25	Search semiallogeneic AND MHC Limits: Publication Date to 1997	15:56:07	58
#21	Search syngeneic and allogeneic MHC Limits: Publication Date to 1997	15:51:45	396
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Date to 1997

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Adoptive transfer of immunity induced by semi-allogeneic hybrid cells, against a murine fibrosarcoma.

Payelle B, Poupon MF, Lespinats G.

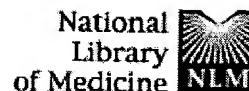
Semi-allogeneic somatic hybrid cells derived from the fusion of a C57BL/6 fibrosarcoma (MCB6-1) and A9 cells (C3H origin) were used to immunize C57BL/6 mice against the parental tumor cells. These hybrid cells expressed histocompatibility antigen of both parental cells (H-2b and H-2k), and failed to produce tumors in normal C57BL/6 mice. A single i.p. injection of hybrid cells induced anti-tumor immunity which could be transferred to normal C57BL/6 recipient mice by immune spleen or peritoneal cells; the effector cells were T cells, as this activity was completely abrogated by treatment with anti-Thy-1.1 antiserum and complement. Among immune splenic T cells, only the light-density T cells, obtained after fractionation on Percoll gradient, were effective in the transfer of immunity. Immunity induced by the hybrid cells was specific for MCB6-1 parental tumor cells. This immunity could be transferred during two brief periods, 7 to 12 days, and 40 to 50 days, after hybrid cell injection; there appeared to be an intermediate period, 12 to 40 days after immunization, during which no immunity could be transferred. These results suggest a suppressive mechanism implicated during hybrid cell immunization and interacting with anti-tumor immune response.

PMID: 6974704 [PubMed - indexed for MEDLINE]

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Expression of two H-2K genes, syngeneic and allogeneic, as a strategy for potentiating immune recognition of tumor cells.

Mandelboim O, Vadai E, Feldman M, Eisenbach L.

Department of Cell Biology, Weizmann Institute of Science, Rehovot, Israel.

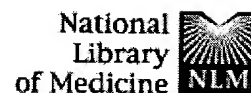
Metastatic clones of some tumors manifest an impaired expression of class I major histocompatibility complex (MHC) antigens. High metastatic, low immunogenic Lewis lung carcinoma clones (C57BL-H-2b origin) express low levels of the H-2Kb MHC antigen. These cells metastasize spontaneously in C57BL/6J mice. Transfection of syngeneic or allogeneic H-2K genes converted such cells to the nonmetastatic state, but did not prevent the growth of the local tumors. Transfection of two H-2K genes, syngeneic and allogeneic, into the highly metastatic clone D122, resulted in reduction of the growth rates of the transfectants and protected the mice from D122 metastases. In contrast, cells transfected with a single class I gene (syngeneic or allogeneic) gave partial protection, or did not protect the mice at all from D122 metastases. The combination of syngeneic and allogeneic genes in the same tumor cell elevated the immunogenic properties of the expressing cells and potentiated the immune response as was demonstrated by in vitro cytotoxicity analysis and by limiting dilution cytotoxicity analysis. Increased immunogenicity by double transfection may constitute an effective therapeutic modality.

PMID: 8750016 [PubMed - indexed for MEDLINE]

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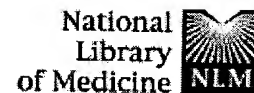
Immunity to B16 melanoma in mice immunized with IL-2-secreting allogeneic mouse fibroblasts expressing melanoma-associated antigens.

Kim TS, Russell SJ, Collins MK, Cohen EP.

Department of Microbiology and Immunology, University of Illinois College of Medicine, Chicago 60680.

Co-presentation of weak tumour-associated antigens along with strongly immunogenic determinants leads to the development of an anti-tumour immune response in recipients syngeneic with the tumour. Tumour immunity develops in mice immunized with tumour cells modified by the introduction of cDNA for interleukin-2 (IL-2). Here, we report the anti-tumour response following immunization with an IL-2-secreting cell construct that expresses tumour-associated antigens, along with allogeneic major histocompatibility antigens. The construct was prepared by transfecting LM(TK-) mouse fibroblasts (H-2D^b) with genomic DNA from B16 melanoma cells syngeneic in C57BL/6J mice (H-2D^b 2b). Transfectants expressing melanoma-associated antigens (MAA) were then infected with an expression-competent retroviral vector containing a cDNA specifying human IL-2. Cytotoxicity toward B16 cells was detected for as long as 5 months in both spleen and macrophage cell populations in C57BL/6J mice immunized with the IL-2-secreting cells. Mice immunized with non-IL-2-secreting, MAA-positive allogeneic cells developed melanoma immunity as well, but to a lesser extent. Immunity to 2 tumour-cell lines expressing the H-2D^b haplotype and to YAC-1 cells was detected in peritoneal macrophages, but not in spleen cells from C57BL/6J mice immunized with the cell construct, indicating that the response to B16 cells was only partially specific. C57BL/6J mice immunized with the IL-2-secreting cell construct survived significantly longer following an injection of viable B16 cells, than mice in various control groups. The contribution of allogeneic antigens to the melanoma immunity was indicated by the failure of mice syngeneic with LM(TK-) cells to develop melanoma immunity following immunization with non-IL-2-secreting, MAA-positive cell constructs. The formation of IL-2 partially compensated for the lack of allogeneic antigens.

PMID: 1533203 [PubMed - indexed for MEDLINE]



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Immunity to melanoma in mice immunized with transfected allogeneic mouse fibroblasts expressing melanoma-associated antigens.

Kim YS, Slomski R, Cohen EP.

Department of Microbiology and Immunology, University of Illinois College of Medicine, Chicago 60680.

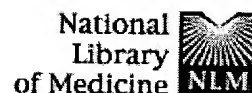
Transfection of genomic DNA from B16 mouse melanoma into LM(TK-) fibroblasts led to the generation of several clones of transfected cells that strongly expressed B16 melanoma-associated antigens (MAA). The transfected cells retained their H-2k markers and served as allogeneic cells with expressivity of MAA in C57BL/6 mice, syngeneic with the melanoma. The cells were capable of eliciting primary anti-B16 immune responses in vitro in spleen cells from C57BL/6 mice. Immunization of C57BL/6 mice with the transfected cells led to the generation of anti-B16 cytotoxic activity in spleen cells, and C57BL/6 mice immunized with the MAA-positive transfected cells were partially resistant to lethal challenge with B16 melanoma cells. Under similar conditions, B16 cells were nonimmunogenic. Therefore, transfected allogeneic LM(TK-) fibroblast cells expressing MAA served as more potent anti-melanoma immunogens than the parental B16 tumor cells themselves.

PMID: 1756533 [PubMed - indexed for MEDLINE]

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Department of Microbiology and Immunology, University of Illinois College of Medicine, Chicago 60680.

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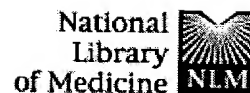
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